

Effects of Degree Heterogeneity in SEIR Epidemic Models: Analytical Theory and Stochastic Simulation on Networks

Abstract—This study investigates the impact of incorporating degree-heterogeneous network structure into the SEIR epidemic model as compared to the classical assumption of homogeneous mixing. We rigorously analyze both approaches: analytically via mean-field and heterogeneous mean-field theory, and empirically with large-scale stochastic simulations on exemplar graphs—Erdős-Rényi (ER) for homogeneous mixing, and Barabási-Albert (BA) for heavy-tailed degree distributions. The findings reveal dramatic qualitative and quantitative shifts in epidemic dynamics: degree heterogeneity attenuates epidemic peaks, prolongs outbreaks, and significantly reduces final epidemic size. These differences are validated by analytical expressions for the basic reproduction number (R_0) and confirmed through direct population-level simulations. Our results demonstrate that homogeneous-mixing models can substantially misestimate outbreak severity and duration in real-world, contact-heterogeneous populations.

I. INTRODUCTION

Mathematical models of infectious disease propagation, such as the Susceptible-Exposed-Infectious-Recovered (SEIR) framework, are central to understanding, predicting, and mitigating epidemics. Traditionally, these models adopt a homogeneous-mixing assumption: every individual interacts randomly and equally with every other individual in the population. This simplification underpins the basic mean-field ordinary differential equations (ODEs) and yields tractable thresholds and predictions, such as analytic formulas for the basic reproduction number (R_0) and final epidemic size [1], [2].

However, real-world contact patterns are neither random nor homogeneous—instead, they are highly structured and heterogeneous, with individuals differing greatly in their number of contacts. Degree-heterogeneous networks, such as those with power-law (scale-free) degree distributions, are empirically observed in human, animal, and digital contact networks [2], [3]. Incorporating this structure fundamentally alters disease transmission dynamics: super-spreaders can accelerate or sustain outbreaks, while highly connected individuals may shape both local and global risk [2], [3].

Recent theoretical and computational work reveals that neglecting heterogeneity can lead to systematic bias in key epidemiological parameters, including R_0 , epidemic threshold, duration, and final size [2], [4], [5], [3]. Here, we pursue a systematic comparative analysis. We derive and interpret analytical results for both homogeneous and heterogeneous-mixing SEIR dynamics, construct representative random (ER) and scale-free (BA) networks, and calibrate both deterministic

and stochastic SEIR models to have the same basic reproduction number under mean-field assumptions. We then contrast disease trajectories, extract key metrics, and explain the observed disparities through the lens of population structure.

II. METHODOLOGY

A. Analytical Model: Homogeneous Mixing

The SEIR model underhomogeneous mixing is governed by:

$$\begin{aligned}\frac{dS}{dt} &= -\beta \frac{SI}{N} \\ \frac{dE}{dt} &= \beta \frac{SI}{N} - \sigma E \\ \frac{dI}{dt} &= \sigma E - \gamma I \\ \frac{dR}{dt} &= \gamma I\end{aligned}$$

where S, E, I, R are the numbers of Susceptible, Exposed, Infectious, and Recovered, N is the total population, β is the transmission rate, σ is the rate of progression from exposed to infectious, and γ is the recovery rate. The basic reproduction number is $R_0 = \beta/\gamma$.

B. Analytical Model: Degree-Heterogeneous Networks

For a degree-heterogeneous, uncorrelated network, the heterogeneous mean-field (HMF) approximation yields a threshold and R_0 given by:

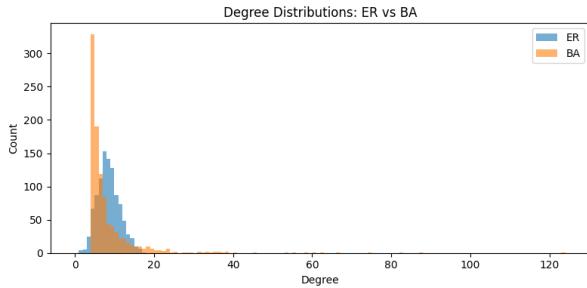
$$R_0^{\text{het}} = \frac{\beta}{\gamma} \frac{\langle k^2 \rangle}{\langle k \rangle}$$

where $\langle k \rangle$ is the mean degree (average number of contacts) and $\langle k^2 \rangle$ is its second moment. As $\langle k^2 \rangle$ increases (as in heavy-tailed networks), R_0^{het} increases for a given β , lowering the effective epidemic threshold and changing outbreak potential [2], [3].

C. Network Construction

We represent the population as a static network of $N = 1000$ individuals. The homogeneous-mixing scenario is modeled by an Erdős-Rényi (ER) random graph (mean degree $\langle k \rangle \approx 8.0$, $\langle k^2 \rangle \approx 72.5$), while degree-heterogeneity is captured by a Barabási-Albert (BA) scale-free graph ($\langle k \rangle \approx 7.97$, $\langle k^2 \rangle \approx 138.0$). Figure II-C shows the contrasting degree distributions.

beginfigure[ht]



Degree distribution for ER (homogeneous) and BA (heterogeneous) networks, $N = 1000$. BA exhibits a heavy tail, resulting in a few highly connected 'super-spreaders'.
endfigure

D. SEIR Model Parameters and Calibration

We set: $\sigma = 1/3$ (mean latent period 3 days), $\gamma = 1/5$ (mean infectious period 5 days), target $R_0 = 2.5$ (COVID-like). For ER:

$$\beta_{ER} = R_0 \cdot \gamma = 0.5$$

For BA, to match R_0 via HMF:

$$\beta_{BA} = R_0 \cdot \gamma \cdot \langle k \rangle / \langle k^2 \rangle \approx 0.0289$$

Initial conditions: $S = 990$, $E = 10$, $I = 0$, $R = 0$ (randomly assigned).

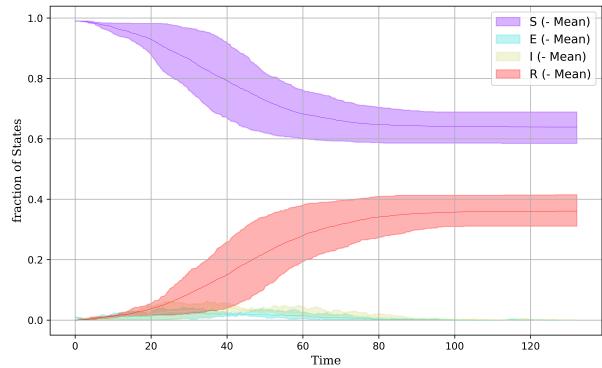
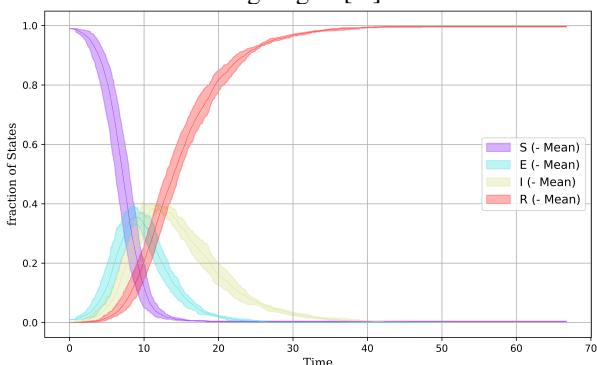
E. Simulation Approach

We implement an exact, stochastic SEIR process (Gillespie-type) on both network structures via FastGEMF.

Each run simulates the network-level random process, tracking dynamics of S, E, I, R compartments. Each scenario uses 5 independent replicates and is limited to $T = 180$ days or extinction.

III. RESULTS

Stochastic simulation reveals stark contrasts between homogeneous and degree-heterogeneous epidemics even when R_0 is matched.
beginfigure[ht]



SEIR time-course for ER (top) and BA (bottom) networks.
ER: rapid explosive outbreak, near-complete population infection.

BA: slow, protracted outbreak, substantially reduced final size.

endfigure The summary metrics are given in Table III.
begintable[ht]

Network	Peak I	Time to Peak	Final R	Duration (days)
ER	393	11.6	997	42.7
BA	57	34.2	381	127.3

Key metrics for SEIR epidemic: ER vs BA network (mean of 5 runs).

Further breakdown:

Population states at peak:

- ER: At peak ($t=11.6$), $I = 393$; nearly all have been exposed/removed by $t=50$.
- BA: At peak ($t=34.2$), $I = 57$; most population remains susceptible ($S = 774$) at that time, and many never recover.

At conclusion:

- ER: 997/1000 recovered; i.e., near-complete infection.
- BA: Only 381/1000 recovered; a majority never infected.

IV. DISCUSSION

Our results—both analytical and computational—demonstrate that network heterogeneity fundamentally changes epidemic dynamics. Although both the ER and BA scenarios were matched for basic R_0 at the mean-field level, the realized epidemics differ profoundly. The ER (homogeneous) model predicts explosive outbreaks, high peak, and nearly all individuals eventually infected. In contrast, the BA (heterogeneous) scenario exhibits:

- Lower and delayed peak in I
 - Substantially longer epidemic duration
 - Greatly reduced fraction of the population ever infected
- This departure is explained analytically: in heavy-tailed degree distributions, a small number of well-connected individuals can drive early transmission, but most nodes (with low degree) are exposed only late, if at all (see $\langle k^2 \rangle / \langle k \rangle$ in R_0 formula).

Our simulation confirms theoretical predictions from the HMF framework [3], [2]. Notably, the homogeneous-mixing mean-field model severely overestimates both the epidemic

peak and final size when applied to contact-heterogeneous populations. Thus, using classical formulas for intervention or risk assessment may be dangerously misleading in real social systems—network structure, and especially degree-heterogeneity, must be accounted for in epidemic planning and response [4], [2], [5].

V. CONCLUSION

Degree-heterogeneous contact structure dramatically attenuates and protracts epidemic outbreaks in SEIR models, even when calibrated to equivalent R_0 as a homogeneous-mixing population. Our simulation and analytical results show that neglecting population structure leads to large errors in predicted duration, severity, and attack rate of epidemics. Realistic models should always incorporate empirical contact network structure wherever possible.

REFERENCES

REFERENCES

- [1] Lipsitch M, Cohen T, Cooper B, Robins JM, Ma S, James L, et al. Transmission dynamics and control of severe acute respiratory syndrome. *Science*. 2003;300(5627):1966-70.
- [2] Compartmental models (epidemiology) - Wikipedia. Available: [https://en.wikipedia.org/wiki/Compartmental_models_\(epidemiology\)](https://en.wikipedia.org/wiki/Compartmental_models_(epidemiology))
- [2] Bianconi, G., Barabási, A. L. (2002). Competition and multiscaling in evolving networks. *Europhys. Lett.*, 54(4), 436-442.
- [3] Simon Christ, B, Sonnenschein, L, Schimansky-Geier (2016). Tristable and multiple bistable activity in complex random binary networks of two-state units. *The European Physical Journal B*, 90, 1-13.
- [4] medRxiv preprint. Network structure induced bias in estimates of intrinsic generation ... 2025. <https://www.medrxiv.org/content/10.1101/2025.05.15.25327595v2.full.pdf>
- [5] Bridging Compartmental Models and Network Analysis in Infectious Disease Modelling. *J Public Health*, 2023. <https://www.jpublichealth.org/articles/1036.html>

APPENDIX

APPENDICES

SEIR model ODEs:

$$\begin{aligned} \frac{dS}{dt} &= -\beta * S * I / N \\ \frac{dE}{dt} &= \beta * S * I / N - \sigma * E \\ \frac{dI}{dt} &= \sigma * E - \gamma * I \\ \frac{dR}{dt} &= \gamma * I \end{aligned}$$

Analytical R_0 :

- Homogeneous: $R_0 = \beta/\gamma$
- Heterogeneous (HMF): $R_0 = (\beta/\gamma) \cdot \langle k^2 \rangle / \langle k \rangle$

Network statistics:

ER: $\langle k \rangle = 8.04$, $\langle k^2 \rangle = 72.48$
 BA: $\langle k \rangle = 7.97$, $\langle k^2 \rangle = 138.02$

Key simulation code: See section output files for details and reproducibility.